Renal blood flow

In a resting adult, the kidneys receive 1.2-1.3 liter of blood per minute, i.e. 21% of the cardiac output.

- **Renal Vascular Arrangement:**
  - The renal arteries are direct branches of the aorta. Each renal artery on entering hilum of the kidney divides to form the interlobar arteries, arcuate arteries and interlobular arteries.
  - The afferent arterioles arise from the interlobular arteries. Each divides into glomerular capillaries in the glomerulus. The capillaries reunite to form the efferent arteriole, which in turn breaks up into the peritubular capillaries that supply the tubule.
  - The capillaries draining the tubules of the cortical nephrons form a peritubular network, whereas the efferent arterioles from the juxtamedullary nephron drain not only into peritubular network, but also into the vasa recta which are hairpin capillary loops that lie side by side with the loops of Henle. The peritubular capillaries reunite to create a venous system consisting of interlobular veins → arcuate veins → interlobar veins→ renal veins. (Fig1-5)

  Therefore, there are two capillary beds associated with each nephron:

1) The glomerular capillary bed: "High pressure bed"

- It receives its blood from the afferent arteriole.
- The hydrostatic pressure in the glomerular capillaries is about 60 mmHg which cause rapid filtration of fluid.
- The pressure in the glomerular capillaries is higher than in other capillary beds due to:

  a) The renal arteries are direct branches of the abdominal aorta.

  b) The afferent arterioles are short, straight branches of the interlobular

arteries.

c) The efferent arterioles have high resistance than the afferent arteriole.

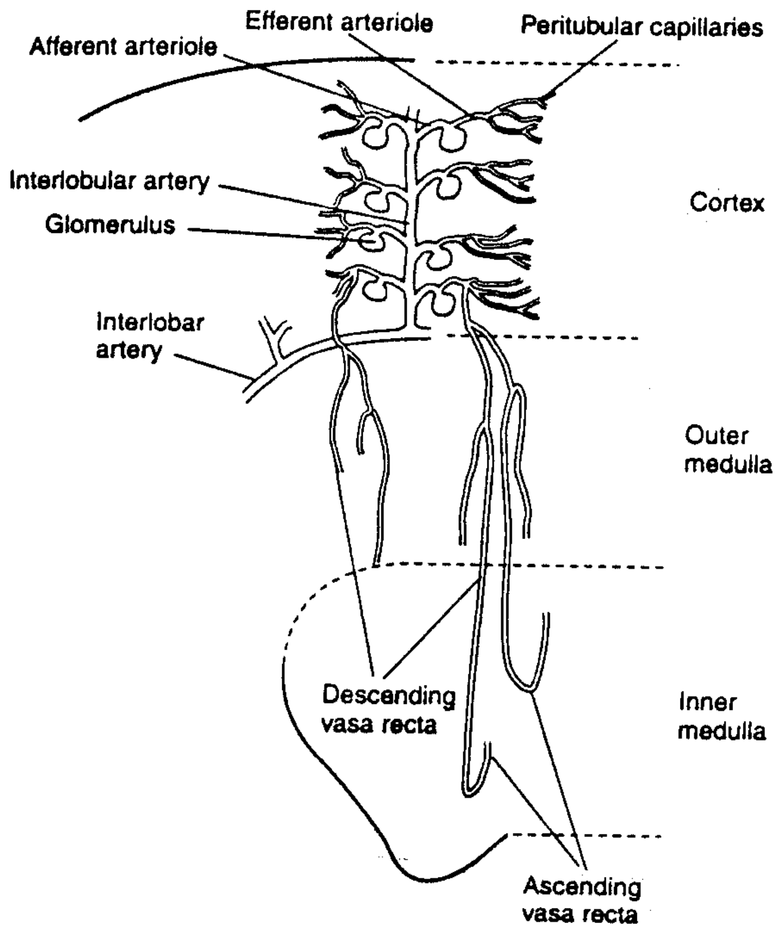


Fig. (1-5): Schematic of relations between bt. Vs. & tubular structures & differences between cortical & juxtamedullary nephrons.

2) The peritubular capillary bed "Low pressure bed"

- The hydrostatic pressure is about 13 mmHg. The peritubular capillaries behave like the venous ends of other capillaries. The low pressure in these capillaries permits fluid reabsorption from the interstitium into the blood.

Regional Blood Flow:

The main function of the renal cortex is filtration of large volumes of plasma through the glomeruli, so it is no surprising that the renal cortex receives most of the renal blood flow (98%) as the renal medullary blood flow accounts for 2 % of the total renal blood flow.

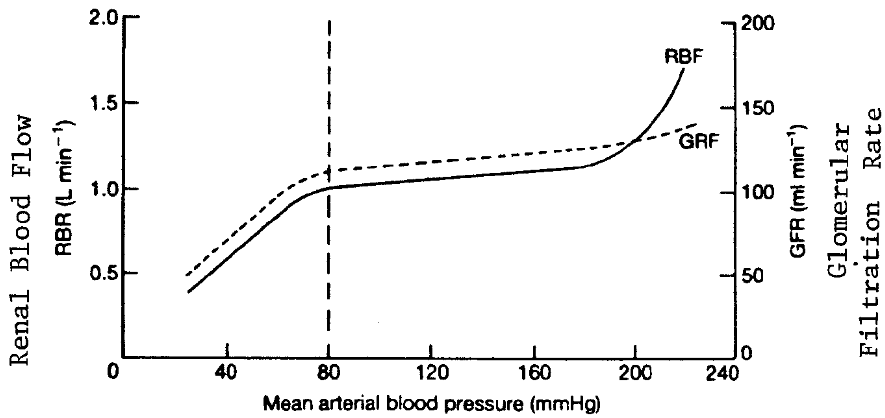
This sluggish blood flow in the renal medulla allows the kidney to form concentrated urine.

Measurement of renal blood flow is discussed later (see plasma clearance of PAH).

Autoregulation of the renal blood flow:

When the kidney is perfused at a moderate pressure (90 – 220 mmHg) the renal blood flow is relatively kept constant by change of the renal vascular resistance (Fig. 1-6).

Renal autoregulation is present in denervated and isolated kidney, i.e. independent of nerves or hormones.



- **Mechanisms of autoregulation of renal blood flow:**

  1- Myogenic mechanism:

a- With rise of pressure: (Up to 220 mmHg).

- Direct contractile response of the muscle of the afferent arteriol due to stretch. This contraction prevents excessive increase in renal blood flow.
- Stretch of the vascular wall increases calcium influx from the

extracellular fluid into the muscle fibers causing them to contract → increase the vascular resistance of different arterioles → maintain renal blood flow.

b-At low pressure: relaxation of the vascular smooth muscles of afferent arterioles → decrease of vascular resistance → maintain a constant blood flow.

2- Tubuloglomerular feedback:

i) When renal arterial pressure increases both RBF and GFR increase. The increase in GFR results in increase delivery of solutes and water to the macula densa.(Fig. 1-7)

- The macula densa responds to increased delivered solute load by secreting the vasoactive substance; adenosine that produces vasoconstriction of afferent arterioles, reduces RBF, HP_{GC} and GFR back to normal.

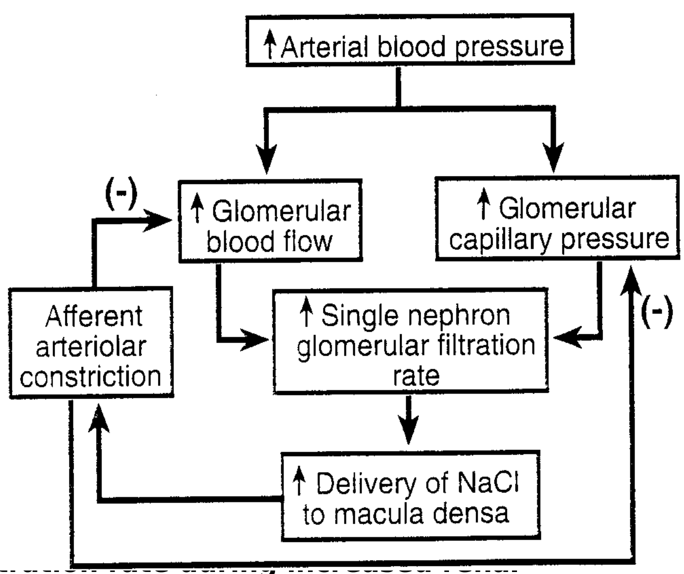


Fig. (1-7): I a f ... **arterial pressure.**

ii) Conversely with drop of arterial blood pressure the HP_{GC} tends to drop. The GFR decreases. Flow rate in the loop of Henle decreases, so that reabsorption of sodium and chloride ions in the ascending loop of Henie increases. Sodium chloride reaching the macula densa decreases. Macula densa sends signals to:

a) Afferent arteriole: poroducing dilatation which raises HP_{GC} and help to return GFR towards normal.

b) Efferent arteriole: producing constriction. This occurs though increase of renin release from JGC. Renin released increases the formation of angiotensin I, which is coverted to angiotensin II. Angiotension II constricts the efferent arteriole, thereby increasing HP_{GC} and return GFR toward normal. (Fig. 2-8).

a and b will increase PH_{GC} and return GFR towards normal. (Fig. 1-8)

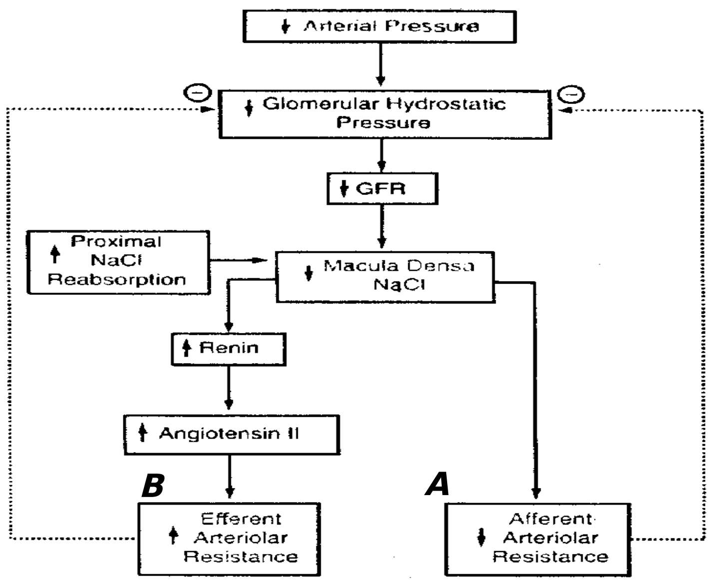


Fig. (1-8): Macula densa feedback mechanism for autoregulation of the glomerular filtration rate during decreased renal arterial pressure.

Aim of autoregulation:

The major function of autoregulation in the kidney is to maintain constant glomerular filtration rate and to allow precise control of renal excretion of water and solutes despite marked changes in arterial blood pressure.

Innervation of the renal vessels and the renal tubule:

Sympathetic fibers supply:

1) **Renal vessels**: Sympathetic stimulation produces vasoconstriction with decrease in renal blood flow and glomerular filtration rate (mediated by α – adrenergic receptor). This occurs during exercise and rising from the supine to the standing position and when the systemic blood pressure fall.

2) **Juxtaglomerular apparatus**: Sympathetic stimulation increases renin secretion by juxtaglomerular cells (mediated by β_1 adrenergic receptors).

3) **Renal Tubule:** Sympathetic stimulation increases Sodium reabsorption by the tubular cells by a direct action of norepinephrine on renal tubular cells. It is mediated via α - or β – adrenergic receptors and it may be mediated by both.

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